bacilli were found, occurring in single elements for the most part but also found in chains of two or more rarely of three elements. No other organisms could be seen. Aerobic and anaerobic cultures were made from the fluid. The aerobic cultures were not sterile but showed no organism resembling the bacillus seen in the fluid. Anaerobic cultures yielded a bacillus resembling that in the bulla and a streptococcus which persistently appeared in the cultures. After some subcultures the bacillus was obtained pure and from its action on animals would appear to have been the cause of the gangrene. Subcutaneous inoculation of one cubic centimetre of a broth culture killed the animal in 24 hours. The guinea-pig soon became ill, had a swelling at the site of inoculation, and showed convulsions of the hind limbs. At the time of death the local swelling had disappeared and there were no sign of injury at the site of inoculation and no emphysema or bullæ. On section, however, a most extensive subcutaneous œdema was found which practically stripped the abdominal skin from the subjacent tissues, which latter were red and infiltrated. In places the tissues were œdematous but there were no gas and no odour of putrefaction. There was much fluid in the tissues which drained into dependent parts. The organs of the chest and abdomen were apparently healthy and there was no peritonitis. The subcutaneous fluid was crowded with bacilli. Anaerobic cultures from the cardiac blood and the subcutaneous fluid yielded growths of the inoculated bacillus, the former in a pure state, the latter contaminated by a streptococcus, as in the original tubes from the bulla. The bacillus is a large, probably motile, Gram-staining, anaerobic organism producing in culture on (1) agar, moderately large greyish-white, rather transparent colonies, with a thickened centre and transparent edge; (2) gelatin, liquefaction with faint turbidity; (3) serum, a thin dry growth; (4) potato, an "invisible" growth; and (5) milk, cultures resembling those produced by bacillus enteritidis sporogenes but without evidence of any copious production of gas. In none of the cultures could any evidence of spore formation be detected.

Cases very similar to the above were reported from this laboratory by Dr. Slater in THE LANCET of July 20th, 1889, p. 108, and by Dr. H. R. D. Spitta in the Transactions of the Pathological Socety, Vol. LIII., Part 2, 1902.

Organisms usually anaerobic have been isolated from many cases of spreading gangrene. Though generally identified as bacillus aerogenes capsulatus, bacillus enteritidis sporogenes, or bacillus œdematis maligni, the organisms isolated rarely conform strictly to the classical descriptions of these microbes. In the present instance the bacillus obtained seems to be most nearly related to the bacillus aerogenes capsulatus though differing in some respects.

Quite recently there has been in the hospital a case of acute spreading gangrene following compound fracture similar to the above. The case was under the care of Mr. Jaffrey and Mr. W. F. Fedden. Gangrene made itself evident 31 hours after the accident and the limb was amputated by Mr. Fedden within 35 hours from the time of the accident, and the man made an excellent recovery. The bacteriology of the case is exactly similar to that already quoted—namely, cultures were obtained of a variety of the bacillus aerogenes capsulatus, and it was impossible to discover any attempt at spore formation.

In conclusion, I have to express my gratitude to Mr. Jaffrey and to Mr. Fedden for allowing me to publish these two cases, and to Dr. Slater for allowing me to make use of his notes on the bacteriology of each case.

A CASE OF ERYTHROMELALGIA.

BY REGINALD G. HANN, M.R.C.S. ENG., L.R.C.P. LOND.

THE majority of recorded cases of erythromelalgia have been, as Weir Mitchell in 1878 originally suggested, intercurrent troubles in the course of disease of the central nervous system. The case now reported must, for the present at least, be considered as idiopathic.

The patient was a married woman, aged 50 years; she had never been pregnant. It seems likely that her occupation as a baker may have been a factor in the causation of her disease, for she practically lived in two rooms, one of which was an unwarmed shop open to the street and the other a very hot room situated over a bakehouse and itself containing a baking-stove in constant use; she was in consequence exposed to very great variations of temperature and was also continually upon her feet.[1] Her previous medical history is a record of many illnesses and numerous ailments. In 1897 she was laid up for eight months as a result of concussion of the brain received in a railway accident. Closely following upon this she was disabled for five months by a painful swelling of the heel, which she insisted very much resembled her present disease. It was too painful to allow her to walk and evidently puzzled her medical attendant, who on more than one occasion desired to incise what he took to be an abscess; she refused to allow it and eventually it cleared up without surgical interference. During the following year she had acute rheumatism and since then many attacks of sciatica, lumbago, and muscular rheumatism. In 1905 she had an acute iritis with adhesions, the cause of which was not apparent. Her present illness began at the end of August, 1906, with pain in the right foot as though she had sprained it. It varied in degree but was not disabling in severity and she continued to work for three weeks when the appearance of a swelling and great increase in the pain drove her to bed. I now saw her for the first time. There was a bright red swelling about the metatarsal region of the right foot to be seen on both dorsal and plantar surfaces; it involved the second toe but not the others. This swelling did not pit and had the light puffy character of an œdematous eyelid; it was the seat of acute pain with a subjective feeling of intense heat, becoming almost unbearable on placing the foot to the ground. The foot was exquisitely tender on the slightest pressure; the superficial veins were distended and prominent; no change of position affected the objective signs of swelling and redness. For two weeks pain of great severity continued without intermission and in consequence she had very little sleep, took food badly, and her general health suffered. Following upon this long attack some improvement appeared, for though a steady, dull ache was present and increased at times to severe pain, still these paroxysms, lasting at first two or three hours and recurring several times a day, soon became less frequent and more bearable; as before the pain was always associated with a subjective feeling of intense heat and was not accompanied by objective changes in the foot. The knee-jerks and plantar reflexes were normal; tactile sensation was not affected. The exciting cause of the exacerbations could not be determined, but any indiscretion such as placing the foot on the floor or allowing it to become too hot in bed, would be sufficient to induce an attack.

As the acuteness of the disease passed away local changes were observed. The swelling, now mahogany-brown in colour, persisted, but was doughy and pitted on pressure and was suggestive of pus formation; to a great extent it had lost its tenderness even to deep pressure, and though the pain was still too severe to permit of walking, it was no longer agonising in severity, nor was it intensified by the dependent position. This stage was most clearly defined during the first week in November after ten weeks of illness. She continued to improve and began to get about the house with the aid of a stick when, early in December, the disease began in the left foot. At this time free desquamation was taking place in the right foot. After a week of pain and a subjective feeling of great cold the left foot became very tender, swollen, and pink, with a subjective feeling of burning. She was again confined to bed. On Dec. 17th she had such pains about her fifth left metacarpal bone that she was obliged to keep her hand in a sling, and although on the following day the pain had gone, on the 23rd, to her great distress, a pink hot swelling with much pain and dilated veins made its appearance. The pain was relieved by placing the arm in a sling but this had no effect upon the swelling or its pink colour. Immediately afterwards a slighter attack occurred in the corresponding part of the right hand. The symmetrical distribution of the lesions was now very striking.

The left foot desquamated during January, 1907; as in the case of the right foot it was not due to local therapeutical applications. From this time the disease became less troublesome. She was able to take short walks and to resume part of her work, though prolonged standing or much exercise gave rise to pain and occasionally to sensations of burning. Objectively there was nothing beyond slight fulness on the dorsum of each foot. This was the condition

[1] Cf. Gerrard (cases in Malays): Dublin Journal of Medical Science, 1904.

still present at the end of April, 1907, and it was interesting to note that during that month she had been complaining of frequent flushings of the face similar to those she had experienced at the menopause. Throughout her illness careful and repeated examination failed to reveal any signs of arterial or central nervous disease.

Treatment was unsatisfactory. The affected limb was always raised. Of local applications glycerine of belladonna with hot fomentations and alternately cold-water packs gave some relief, provided that extremes of heat or cold were avoided. Internally aspirin, salicylate of sodium, ergot, and calcium chloride all in turn seemed to do good when first ordered, but were soon given up as useless.

This patient showed three clearly defined stages of the disease: (1) A premonitory one of severe pain, now better, now worse, without swelling or redness; (2) an acute stage of the severest pain, worse in the dependent position, with a tender, pink, puffy swelling; and (3) a chronic condition with slighter pain except on using the limb (attacks no longer spontaneous), with tenderness only on deep pressure, and a brown-red swelling. In the hands this last stage was represented by slight tenderness without swelling or discolouration.

Some points in the case may be briefly noticed. Unlike the cases recorded by Collier,[2] in which the erythromelalgia was a symptom-complex in the course of disease of the central nervous system (tabes, insular sclerosis, traumatic neurasthenia, &c.), in my case pain always preceded the appearance of vaso-motor (objective) signs. The length of the first attack—two weeks—is unusual, the attacks individually generally lasting for a few hours. Desquamation is certainly rare; it occurred in a case under Voorheis.[3] At no time did the discolouration suggest the blueness of venous obstruction. The flushings of the face afford another indication of the instability of the patient's vaso-motor apparatus. Varying degrees of generalisation of the vaso-motor or sensory symptoms of the disease have been not uncommonly reported; one of Weber's patients suffered habitually from hyperæmia of the face and tongue.[4]

The case impressed one with a feeling of the ease with which erythromelalgia, and especially less typical instances of it, could be overlooked in the course of general practice.

Leeds.

Medical Societies.

ROYAL SOCIETY OF MEDICINE.

PATHOLOGICAL SECTION.

President's Address.—Fatty Degeneration of the Blood.—Macroglossia Neurofibromatosa.—Imperfect Hermaphroditism.

A MEETING of this section was held on Oct. 15th, Mr. S. G. SHATTOCK, the President, being in the chair.

The PRESIDENT, after making some historical allusions to the evolution of pathological science, observed that it was a favourite belief of his that there would always be a new disease for anyone to discover who seriously undertook the task of differentiating it from the number of undiagnosable and obscure conditions, both clinical and pathological, which so frequently obtruded themselves into notice. Their present conception of pathology was that of a science which must be studied from every side, for additions to it had not infrequently been made from wholly unexpected quarters. They could not recognise a pathology from which clinical observation and physiology any more than botany and zoology and physics and chemistry were excluded. In London, possessed as it was of so vast an amount of clinical material, it was especially fitting to investigate disease in its more immediate relation to medicine; together pathology and medicine might be studied with the highest mutual advantage; to divorce them would be disastrous to the progress of both.

The PRESIDENT and Mr. L. S. DUDGEON made a communication upon Fatty Degeneration of the Blood. One peculiarity in the constitution of the blood whereby it differed from other tissues was the fact that although its cells, as in the more stable forms, undergo division, both mitotic and amitotic, and had in this sense a life of their own, yet the primary sources of its elements were the blood-forming organs. The blood degeneration which had been as yet most studied (other than the so-called polychromatophilia of the red cells) was that usually regarded as glycogenic and consisted in the abnormal—i.e., excessive—appearance of glycogen, chiefly in the finely granular polymorphonuclear leucocytes, although granules occurred also in the eosinophile or coarsely granular polymorphonuclear cells, and to a lesser degree in the lymphocytes. If pathological effusions, such as those of pleurisy and peritonitis and purulent exudations, were excepted, the process of fatty degeneration had hitherto been looked for and studied only in the solid tissues. Yet it would be apparent on theoretical grounds that in cases where fatty degeneration of the last-named tissues was due to a deficient oxygen-carrying power of the blood, or to a toxic condition of it, the state of the blood might react upon itself so as to induce similar changes in its own leucocytes. Without entering into the chemical question as to the source of the fat in fatty degeneration, how far, that is, it was due to a transformation of the proteid of the cell and how far to the accumulation of fat reaching the cell from without and appearing in it because not utilised, the cardinal difference between the two processes remained, even though the source of the fat should be the same in both. In the one case the fat accumulated or was stored in a cell of which the cytoplasm was uninjured; in the other it appeared for the reason that the latter was damaged and rendered incapable of utilising the fat which reached it. With the fat that was not free but combined in the cell neither term dealt; the presence of this was not recognisable either by its microphysical or microchemical properties. At the outset it was obviously essential to exclude the use of fat solvents, like absolute alcohol and ether, as fixing reagents, and essential also that the blood should not be dried. The method finally chosen was that of making films on slides and cover glasses. The film having been made on the slide in the usual manner the latter was immediately placed with the film downwards in a specially-devised chamber of formol vapour. After not less than 15 minutes (though the films might be left in the vapour for 24 hours or longer) the slide was removed and at once placed in the vertical position in a glass of "Scharlach" for from 24 to 48 hours. Hæmalum was used afterwards as a cell stain. Their examination of blood films extended altogether to 79 cases and comprised various morbid conditions. Nevertheless, it was in comparatively few that the President and Mr. Dudgeon satisfied themselves of the presence of fat in the leucocytes. Fat was found in the following cases: chlorosis, chronic Bright's disease with anæmia of chlorotic type, carcinoma of the pylorus with anæmia of chlorotic type, influenza, pleurisy with pericarditis, toxæmia of pregnancy, acute pneumonia, purpura, diabetes with lipæmia, myelæmia, lymphadenoma, and acute cerebro-spinal meningitis. If these cases were analysed it would be found that most of them came in the category of toxic diseases, some of an acute and others of a chronic kind. In regard to the toxic conditions the President and Mr. Dudgeon believed that the fatty change present in the finely granular polymorphonuclear leucocytes resulted from the direct action of the toxic substances present in the blood itself; that is to say, that the change was one of proper fatty degeneration as distinguished from the ingestion or storage of fat by a healthy cell. The fineness of the fatty points, their varying number in different cells in the same film, and the fact that they occurred in abundance in cases where the condition of the patient excluded the possibility of so large an amount of fat being merely ingested from the blood by the circulating leucocytes all pointed to that conclusion. Thus in the three cases of acute pneumonia the blood was taken in one on the sixth day two hours after the crisis; in another on the fifth or sixth day before the crisis; and in the third five hours after the crisis. Furthermore, the fact that in such an acute disease fatty degeneration actually did occur in the renal epithelium and cardiac muscle proved the ability of the toxins to effect such damage also upon the circulating leucocytes. That the degeneration of the leucocytes had occurred in the blood and not in the bone marrow might be held as almost certain. It might have been thought, *a priori*, that the presence of fat in the finely granular polymorphonuclear leucocytes would be a sign of grave significance as indicating not only a high degree of toxæmia but an interference with the

[2] Collier: THE LANCET, August 13th, 1898, p. 401.
[3] Voorheis: Medical News, New York, 1904.
[4] British Journal of Dermatology, February, 1904.